this condition, and between them, foreign surgeons particularly, are much divided. The first operation is that of immediate drainage, while the second consists in exposing the liver, allowing it to become adherent to the abdominal wall, and then opening it twenty-four or forty-eight hours later. In the hands of an experienced surgeon the former operation would certainly seem to be the most satisfactory procedure. Under doubtful aseptic circumstances an inexperienced operator would probably be wise to follow the second method. Smits[1] reports, from the island of Batavia, the most gratifying results from the employment of the two-stage operation, 16 recoveries out of 19 operations. It is doubted, however, that these good results were altogether due to the two-stage operation. After the location of the pus by the needle we believe that there is no better instrument for incising the liver tissue over the abscess than the Paquelin cautery. Curettement of the abscess wall is a measure strongly advocated by Fontan, but its employment in abscesses of short duration would seem questionable and, in fact, the present case would tend to show that it is not an essential part of the treatment even in abscesses of long duration. In the introduction of the drainage-tube care must be taken that the end of the tube does not rest on the abscess wall, else such pressure might produce necrosis and perforation at this point. One point in the technique of this operation which is frequently dwelt upon is the stitching of the liver to the abdominal wall. This procedure does not seem necessary if the precaution is taken to carefully surround the tube with gauze, and might possibly interfere with the subsequent contraction of the abscess cavity. It was not done in the present case nor in another recently seen in which an equally good result was obtained.

A STUDY OF SOME CIRRHOSES OF THE LIVER.

By Beverley Robinson, M.D.,
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No doubt the majority of practitioners believe they know pretty well about cirrhosis of the liver. It is a common affection, particularly in hospitals and dispensaries. In its advanced stages it is readily diagnosed, as a rule.

In regard to its prognosis and treatment: These appear relatively simple in that formerly at least it was assumed by very many that we had to do with a fatal complaint; and as for remedial measures, they resolved themselves into few, among which paracentesis at the last stage only was imperative, mainly to relieve distress. How far such notions to-day are removed from truth I shall endeavor to show later.

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[1] New York Medical Journal, February 9, 1901.

That the liver is a difficult organ to study and know accurately few will deny whose opportunities and mental make-up prove them competent observers. As compared with other abdominal or thoracic organs, where does it stand? The lungs can be inspected, palpated, percussed, auscultated, and in a way measured. So, perhaps, can the liver; but in the former case we seem to be in more accurate touch latterly by reason of sputa examinations under the microscope and inoculation experiments. The heart and its disorders have been the source of so many investigations in the physiological laboratory, so many accurate clinical observations in the office and the class room, hospitals, and dispensaries—everywhere and at all times—that little new may be added. With the advent of physiological chemistry, the use of the microscope, and the opportunity, daily or hourly almost, of knowing precisely what the urine shows, the kidneys as organs are very clear to us. With the liver, on the contrary, much seems still conjecture and hypothesis. We speak of its torpidity, engorgement, anæmia, its functional derangements, its organic lesions, with glibness at times; and yet there is much that is obscure about its functions, healthy and morbid, that is by no means evident, and about which there are very honest and almost irreconcilable differences.

In this connection, only very lately, Dr. Goodhart writes: "The largest organ of the body, its imports and exports must be enormous, and from the familiar way in which it is spoken of there cannot be a man in the whole world who does not think he knows all about it. But what are the facts? We know something about the physiology of the liver; but this knowledge has been mostly obtained by experimentation on the lower animals, by observations that occasional cases of disease afford us, and by certain inferences that we draw—very much at second hand—from the changes produced by disease in the organ. But all these things—valuable as they are, and without which where we should be I do not know—yet are very far from giving us that real and intimate knowledge of the living organ that we require to enable us to treat its diseases."[1]

Should this not be adequate reason why we approach the study of the cirrhoses with much diffidence and full recognition of our deficiencies, even in the rôle of an upright reporter? "Good men and true" have essayed this work faithfully in the past, and told us many things we should know. A great deal more is required, much research remains to be done, and only by degrees may we legitimately hope to secure truth—entire and with full details.

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[1] British Medical Journal, August 3, 1901, p. 251.

In what relates to the cirrhoses there are many undetermined questions. The views ordinarily held are not entirely correct. In the first place, the clinical facts, the more we become conversant with them, do not justify invariably the gloomy prognosis of these affections which has been entertained. Instead of separating with discrimination, there has been confusion, because all forms of the disease have been thrown together and included as one. In fact, here as elsewhere, the work of the dead-house has reigned supreme for a time, and the close watching of patients during life has been deemed of lesser value, when it should always, as I believe, be the first thought of every good physician. How, indeed, can we treat a patient properly unless we know just what symptoms he presents during life and in what manner and to what degree function was disturbed? Everyone who has grown gray in harness and whose experience has widened and deepened knows that the morgue and the laboratory by themselves ignore too much and too often the intricate and obscure of our economy in a dynamic sense, and hence must be always controlled, as it were, from the horse-sense point of view of even the humbler humdrum daily worker at the bedside.[1]

There are different forms and degrees of cirrhosis. Not all at once and every time does the final atrophic stage, with its irremediable anatomical conditions and its absolutely gloomy horoscope, show itself. This form is slow and insidious of development. At first it is not recognized; there are few or no symptoms we can attach to it. At best we can only be suspicious.[2] If we have to do with a chronically hard drinker of beer or spirits, of course, we think of the liver and fibroid changes; and then if there be palpitations of the heart, evidences of dyspepsia, vascular stigmata on the face, corpulency, and a somewhat enlarged liver we cry a halt to bad habits, and the outlook may be sombre. On the same lines, if we find loss of strength, inappetence, and a failure of nutrition, as shown by loss of weight, even though the liver be of normal size apparently, we cannot avoid somewhat gloomy forebodings unless we are able soon to control habits and regimen. But are we sure—may we be reasonably certain—with such vague characters that we shall even later be able to fix an absolute and correct diagnosis? The answer—the only one—must be negative. The sooner the better for every medical student to know that disease does not run closely at any time along so-called prescribed lines. It varies, it differs; one day we see certain forms of disease, another day it is just the contrary. All cases are in a certain sense individual, personal. There is nothing wholly general and all-absorbing about any one instance. Take, for example, the abdominal effusion in cirrhosis—the ascites which in the later stages is so striking, so characteristic, so grave of import in the eyes of many that it seems like to a funeral knell in its fatality. Is it so? May it not appear soon, and in its earlier stages may it not be treated wisely and advantageously? And may not a patient do well for a long while, thus treated? And why is this? May not very many of the liver cells still be functionally healthy, quite capable of carrying on good nutrition? Is not fibrous growth in the liver usually slow? Does it not leave many lobules untouched for many a day by its contracting power, and otherwise in what degree is it really pernicious? These and other queries immediately arise to one's mind. It is, moreover, a fact of daily experience almost to have patients come to our office or to out-door clinics in whom ascites is undetermined. We are unable to affirm positively that it exists. And this is true not for one examination only, but for many. Days, months, and even years elapse with some patients before we can say convincedly this *is* or this is *not* a case of hepatic cirrhosis.

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[1] Vide Andrew Clark, British Medical Journal, February 3, 1883, p. 191: "Address on Clinical Investigation before the Clinical Society of London."

[2] In writing of early stages of hepatic cirrhosis, Billings claims there is a relatively large number of patients whose symptoms are those of neurasthenia, myalgia, mononeuritis, or gastro-intestinal disturbance.—Medical News, July 26, 1902, p. 167.

The three principal forms of cirrhosis which are recognized by writers are (1) atrophic, (2) hypertrophic, (3) syphilitic. Besides these there are so-called minor, even undetermined, forms. Usually the latter are due to passive congestion, brought on by pressure from neighboring tumors or indicative of some chronic disturbance of heart power, structure, or action. In these instances, especially where the heart is the primary cause, the abdominal ascites is merely a symptom which is part of a general anasarca. It may be, however, that the ascites has something special in it; it may stand by itself, as it were, so far as effusion is concerned in the serous cavities. Then we have to do with an expression of cardiac inefficiency accompanied by one of the known effects of portal obstruction due to fibrosis around the vessels of the liver. In these cases we should look rather for hypertrophy than atrophy. The liver is enlarged and engorged. It is filled with blood from overcongestion. There may or may not be already marked fatty change, and thus, instead of the dense, hard, tough liver with sharp lower margin, which is said to characterize the cirrhotic liver, we may have a greasy, rather soft structure, which leaves the impress of the finger upon its surface when we press with even very moderate force. While, then, it may be admitted, and is certainly true with limitations, that all three forms of cirrhosis of the liver are characterized by increase of fibrous tissue, which penetrates its structure and distributes in somewhat different ways along the branches of the interstitial tree, it is also true that these forms are all different in many particulars.

Their pathology, to begin with, is not the same. In one form—the hypertrophic—the fibrous tissue is finer, less formed, accompanied by a cellular growth, and runs along the smaller branches interstitially. In the atrophic form this fibrous tissue is coarser, better formed, shows greater tendency to contract upon and disorganize liver cells, extends along larger branches of the interstitial tree, and is not accompanied by cellular growth to the same degree at all.

In connection with the increase of connective tissue it has been proven experimentally that the action of alcohol on the liver of animals is also to cause granular and fatty degeneration of the liver cells. According to Vaughan, the increase of the connective tissue is small in amount, and only reported by some observers. Vaughan concludes that "the connective tissue changes are subsequent to and dependent upon alteration in the hepatic cells."[1] Later on he writes that with the clinical and experimental evidence hitherto obtained we are justified in believing "that in alcoholic cirrhosis the pathological changes begin in the hepatic cells."

From what has been said concerning the pathology and etiology of atrophic and hypertrophic cirrhosis it has been shown that the two forms may exist as types. They may also be present combined, as it were, in the same individual and exist as one disease. The atrophic form is clearly due, as a rule, to alcohol; the hypertrophic to infection (Vaughan); but the infection may precede or follow the toxic poisoning, and the result is a mixed form of cirrhosis. These forms, both clinically and at the autopsy, are very difficult of precise diagnosis, so intimately and curiously combined are their symptoms or lesions.[2]

In the syphilitic cirrhosis we have those broad bands of fibrous tissue which penetrate between the lobules and draw the liver down near the surface in such manner as to leave the puckered, scarred appearance, with bossy, prominent, irregular masses between which are so characteristic.

As to the course and clinical symptoms: These, we shall see, differ widely and obviously in many ways. In atrophic cirrhosis, for example, the main distinguishing clinical feature, as we could almost premise from its pathology, is the evidence of marked portal obstruction—at least this is true almost as soon as the disease is clearly determined. Hence arises the abdominal effusion, and with this most significant and almost portentous sign we have hemorrhages from the stomach and bowels. Sometimes these are small and infrequent, and give no great and immediate alarm if their quantity alone is considered; but considered from the point of view of their evident causation, great apprehension is invariably excited, and justly so. Shortly we may have, and do often, those abundant and depressing blood losses so difficult to arrest, and which point to the urgent necessity, and none too soon, of employing all rationally known measures to ease up an obviously crippled organ and to restore falling strength. How unfortunate it is in such cases if the somewhat too enthusiastic surgeon finds indications for abdominal exploration or an operation for internal hemorrhoids! Jaundice is a rare sign in these cases[1]—at least real jaundice, with urine loaded with bile pigment and acholic stools. Of course, the dull, earthy hue of the skin and the subicteric tint of the sclerotics are frequent phenomena which serve in a measure to fix our diagnosis. Sometimes "adhesive pyelophlebitis closely resembles the atrophic form of interstitial hepatitis. The rapidity with which the peritoneal effusion reforms after tapping is an important diagnostic sign."[2]

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[1] Journal of American Medical Association, October 5, 1901, pp. 878, 879.

[2] In a patient I saw last summer, who subsequently died, the liver was stated during life to be *much enlarged* by several very competent observers—both surgeons and physicians. I was able to satisfy myself after death, by intra-abdominal digital exploration, that the liver was *not notably* enlarged. I was also convinced that an intra-abdominal mass, in close juxtaposition with the lower margin of the liver, gave rise to an error of diagnosis in this regard, although the case was proven not to be one of cirrhosis of the liver.

These cases of atrophic cirrhosis in reality are long as to duration. They appear short merely because they are unrecognized prior to the advent of the ascites, and then the inevitably fatal termination seems to stand out imminently, since a few months or a briefer period may bring about a fatal ending of the case.

The first and imperative thing to do with these patients is to lay down the law which forbids alcoholic stimulation in any form, because, as we know, in the chronic irritation thus produced we find the immediate and efficient cause of the majority of such cases. Of course, there are degrees of injurious action. The man who has drunk heavy beers for a long while and in large quantities, combined, it may be at times, with daily potations of spirits undiluted and containing fusel oil, is the worst type with which we have to do. Such an one is the longshoreman, hodcarrier, and other laboring man, or the chronic loafer who comes to our out-door clinics or to our hospital ward, when, on investigation, we find the contracted, small hobnailed liver, which shows the condition of multilobular cirrhosis and also many accompanying pathological changes. The spleen and pancreas are often hard and functionally decrepit, the kidneys show marked interstitial changes, the stomach is thickened, atrophied, and congested; but, above all, the heart is weak, flabby, degenerated. Fibrous myocarditis is sometimes made out at the autopsy; more frequently we have more or less fatty change, apparent to the eye and corroborated with the microscope. The general vascular system does not escape, and capillary arteriofibrosis has run its course, so that there is scarcely a sound arterial coat to be found, no matter what organ we turn to. This is, indeed, a gloomy and, alas! too true a picture. Alongside, fortunately, we have another far more hopeful. The man-about-town, the broker, the business man—even the lawyer and the not too wise physician—whose drinking consists in two or three or more "Manhattan" or "Martigny" cocktails every day, taken upon an empty stomach and as "a pick-me-up," as the saying goes; the genial, pleasant fellow who gives himself a rest of an evening, and who frequents the club and the billiard-room, and who before the evening is gone has consumed several drinks of "rye" or "Scotch" or "unsweetened," is also an apt candidate for admission among the cirrhoses of the atrophic order. Fortunately, such an one drinks good spirits, as a rule, in the sense that it is relatively pure and unadulterated. Moreover, his food is well cooked and assimilable. He bathes, and is therefore cleanly and has an active skin; and with a healthier body, perhaps, to start with he does not suffer so soon or so hopelessly as the poor chap I have previously considered. Moreover, when the first premonitory signs of disease occur, if he be even moderately wise, he consults his family physician, when I would fain believe he secures a mental and moral shaking up, with which a little useful medicine added thereto allows him to guard more carefully his still useful liver cells, and gives declared fibrosis an opportunity to cease development and extension for a time.

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[1] Where fatty degeneration and cirrhosis coexist profound, persistent jaundice is less infrequent. A patient thus affected, as shown by the autopsy, died in one of my wards at St. Luke's Hospital May 8, 1902.

[2] James H. Wilson, Medical Record, December 7, 1901, p. 909.

Without going further in this description I trust I have been able to show and bring home to my readers what they all know, what they all feel and see around them every day. Turn, now, to our hypertrophic form of cirrhosis, and what may we note? Here, instead of atrophy and contracture and loss of size, we have enlargement and increase of weight. In addition, there is to be found a singular development of new biliary canaliculi on the periphery of the lobules, which is so distinctly shown in sections for microscopic insight. This latter division is the one so ably described by Hanot, Charcot, and other French writers, and about which there is such wide divergence of opinion elsewhere. Suffice it to add that in typical forms these must be very rare, infrequent cases. For my own part, I do not recall to have seen and observed a typical one—at all events, not one where the post-mortem researches justified altogether a previous clinical diagnosis. In the hypertrophic cirrhosis of the sort we do see from time to time the fibrosis as it extends into the liver seems to mark for its own a few cells here and there throughout the structure, or certain individual lobules. In this hypertrophic form ascites does not often occur, nor should we expect it if we pay due regard to the special characters of the fibrosis formation. Jaundice, on the contrary, is a marked and almost omnipresent symptom; and it is the real jaundice—not the aborted state, so to speak—to which I have referred in the atrophic form of cirrhosis, *i. e.*, icteroid hue.

Like to atrophic cirrhosis, hæmatemesis is not infrequent, and, when it occurs, abundant and very threatening. Many of these hemorrhages in cirrhosis originate at the lower end of the œsophagus. They are often confounded with hemorrhages which come from ulcer of the stomach.[1] In either case, but especially the former, we should be very chary about the introduction of a stomach-tube, as a dangerous or even fatal loss of blood might take place immediately. With the hemorrhage from the œsophagus or stomach we see developed many acute symptoms. There is fever, and often the temperature ranges high. There is delirium of an active kind, pointing to a toxæmia and blood infection. "Chills and sweating are not common" in the hypertrophic form of interstitial hepatitis, "thus serving to differentiate it from impacted gallstones." (Wilson.)

Later the patient may relapse into coma from which there is no awakening. Before these symptoms of delirium and coma become manifest we usually have several repeated intermittent attacks of pain, with fever. The pain is localized over the hypochondrium, and the liver is tender.

In these particulars we have an approach to the syphilitic form, and except for the history, the different physical signs, and other symptoms, we might have a difficult diagnosis; but this is not true for the careful diagnostician, who also considers the ordinarily chronic course of the disease.

As to causation, it is obscure. Frequently the spleen also being enlarged a malarial origin is suspected. There are too few observations accurately made in which the plasmodium has been discovered to altogether justify this opinion. I rather believe that alcohol is here again a more probable and efficient factor in bringing on this change in liver structure. One statement would seem to be borne out by facts that we do know, and it is that the obstruction of the bile ducts by fibrous tissue seems to account for the genesis of the acute symptoms which mark the course of the disease and point to inflammatory conditions, more or less lasting. According to some authors, such inflammatory conditions are wholly ignored, and they affirm that an infective process is the sole, undeniable cause of hypertrophic cirrhosis. In this conection Vaughan[1] writes: "With our present knowledge it must be attributed in all cases to infection." If this be admitted, pathologically, it may be stated that "the epithelium of the gall ducts is the site of the primary involvement." (Vaughan.)

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[1] According to Knapp (Medical Record, March 1, 1902, p. 334) these hemorrhages are sometimes erroneously "diagnosed as pulmonary tuberculosis," when they should be considered "cases of ulcer of the œsophagus."

According to Bouchard (Revue de Médecine, October, 1902) hemorrhages in cirrhosis, which occur outside the territory of the portal vein, are in relation with pre-existing arterial lesions, affecting various organs, and do not depend upon abdominal plethora. The arterial lesions are chronic degenerative changes probably due to arterio-sclerosis.—Boston Medical and Surgical Journal, September 11, 1902.

In the syphilitic liver we find marked induration, at times fairly recognizable by our tactile sensations. The liver itself is, as a rule, larger than normal. This is not an invariable rule, and just the contrary may be found, no doubt, in those instances where the fibrous contracting bands are broad and numerous. The gummata on the surface of the liver when they exist are often easily recognizable. Of course, we may confound them with malignant nodules on the one hand or perhaps the hobnailed condition of atrophic cirrhosis on the other; but the bossy, lobular forms of the tumors, together with the previous history and other signs of syphilis, or, in the absence of both, the notable cachexia of cancer, if present, enable us to be pretty confident as to our differential diagnosis. In reasonable doubt an accurate blood count with differentiation of white cells will help us much. A decided and relative increase of lymphocytes and of eosinophile cells, according to Neusser or Cabot, would make this point very valuable. Of course, "a disappearance of the tumor after a tentative antisyphilitic treatment argues most positively in favor of gumma."[2]

So far as clinical symptoms go, syphilitic cirrhosis resembles one of the other forms. There may be jaundice and no ascites; there may be both ascites and jaundice.[3] On the other hand, there are instances in which neither sign is present. At an advanced stage, however, ascites can usually be determined; and what is of particular value is the localized pain in the right hypochondrium which is usually present. These pains are not constant, as a rule, but come on intermittently, or at all events are greatly intensified if previously they have been present in a mild, dull form. The pains during the exacerbations may become so intense as almost to resemble those of gallstones. If the disease has existed for a long while there is decided loss of weight, but scarcely ever to the degree which characterizes malignant disease. Constipation and various functional disturbances of the stomach and intestines are frequently present.

The congenital form of syphilis of the liver I do not remember to have met with; if I have my memory is too uncertain to be a faithful reporter.[4] By those who have seen or described it it is said to be a diffuse fibrosis, where the abnormal tissue formation penetrates into the lobules or between the cells. The fibrous tissue is an immature development. The liver itself is large and it is accompanied by jaundice, ascites, and emaciation.[1] While as regards the pure pathology of cirrhosis, as Cheadle says, there is more or less unanimity among writers, this is not true of the causation of symptoms as connected with morbid anatomy, nor of the course and clinical manifestations of the disease in its various forms. One of the questions for settlement is to know whether the atrophic form is anything more than a later stage of the hypertrophic. Further, more definite judgment as to Hanot's biliary cirrhosis is desired, and as to whether hypertrophic cirrhosis is a real entity. These and many others are of singular interest. It is ordinarily accepted that atrophic cirrhosis is mainly caused by alcohol; indeed, that it is the essential cause in the vast number of cases. Still, malaria has seemed to be a sinning factor at times. Unfortunately, a typical case of malarial origin is rarely found, at least with us in New York. This may be accounted for, and doubtless is, by the relatively mild form of malaria from which we suffer. The æstivo-autumnal sort, with the characteristic crescents in the blood, does not occur often. They are relatively exceptional. Now, it is in just these cases or those of long-continued malarial cachexia, where we find great enlargement with fibroid induration of the spleen and also of other abdominal organs, notably the kidneys. Why, then, should we anticipate finding well-marked morbid effects of this sort in the liver if, as I do believe, the liver as an organ is not nearly so apt to be much affected by malarial poison as is the spleen? Also, I would state that it is most unusual for us to discover cases of profound malarial poisoning where we could absolutely eliminate the disastrous influence of alcohol from the equation; and yet it is stated, even by those who claim never to have seen a case of malarial cirrhosis, that there is nothing absolutely improbable in the assumption, inasmuch as malarial indurated spleen is fully determined. To my mind, this reasoning by analogy is very lame and inapplicable to the liver, in view of the fact of its very different structure and functions. When we consider how prone the liver is to become engorged and somewhat cirrhotic in all cardiac affections as well as in all obstructive lung diseases, it would seemingly be doubly reasonable to eliminate the malarial factor in causation, unless pretty clearly evident. I direct particular attention to this statement, because I know that in most text-books malaria is insisted upon as a cause of atrophic cirrhosis of the liver. Osler,[1] in this connection, writes: "In our large experience with malaria during the past nine years not a single case of advanced cirrhosis due to this cause has been seen in the wards or autopsy-room of the Johns Hopkins Hospital." Now, it is well known that the malarial manifestations found in the city of Baltimore—a few indigenous, many coming from regions farther South—are more virulent than those we meet with in New York or other cities of the North in the United States.

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[1] Ibid. cit.

[2] Einhorn, Medical Record, August 17, 1901.

[3] Stockton states when jaundice occurs, even with apparent alcoholic cirrhosis, especially if accompanied by cachexia, it is well to suspect syphilis.—Medical News, June 28, 1902, p. 1244.

[4] According to Osler it "is rare and nearly always overlooked."

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[1] Vide Cheadle. Lumleian Lectures for 1900. It affords me great pleasure in this connection to speak with admiration of these lectures, and to state that I have freely used them in writing this paper. Indeed, I have followed his outlines (Cheadle's) because I could find none better.

Since the above was written, Dr. George G. Sears and Dr. F. T. Lord have published in the Boston Medical and Surgical Journal of September 11, 1902, a very important paper on cirrhosis of the liver, based on all the fatal cases in the Boston City Hospital and the Massachusetts Hospital since 1896 in which the diagnosis was proved by histological examination. Many of their conclusions, according to the London Lancet of October 15, 1902, are only confirmatory of those published in England by Dr. Cheadle and by Dr. Hale White.

Osler also writes,[2] in speaking of the liver: "Only those cases in which the history of chronic malaria is definite, and in which the melanosis of both liver and spleen coexist, should be regarded as of paludal origin."

There seems to be little doubt, also, that hypertrophic and atrophic *alcoholic cirrhosis* are distinct forms of disease and not different stages of the same form. This affirmation seems reasonable when we consider the very marked difference in the distribution of the fibrosis as well as of its nature in the two cases. In the one the affection is markedly multilobular; in the other it is monolobular in quite as pronounced degree. According to Cheadle, the error in confounding the two diseases arose originally from the hypothesis that Hanot's so-called biliary cirrhosis was simply the hypertrophic form. In many instances of cirrhosis of the latter form there is unquestionably a period when the liver is simply congested and enlarged, and yet no formation of abnormal fibrous tissue is yet begun, not to speak of any degree of contracture which later on may prevail. In these cases the liver is said to be much softer than it is where hypertrophic cirrhosis has become developed. This statement is no doubt true, and can be shown obviously in the autopsy-room on rare occasions. When it comes to actual clinical work and interpretation I am obliged to confess that this stated hardness as compared with softness, where the large liver is merely congested, is very difficult to differentiate. I have seen many cases of enlarged, smooth liver where there were few or no symptoms of ill health, unless, perhaps, slight evidences of dyspepsia should pass for such. In these cases the tactile sensations, so far as relative hardness is concerned, revealed little or nothing from this standpoint. This judgment is specially correct, I am confident, where the patient is corpulent, with thick abdominal walls, due to the deposit of adipose tissue. In spare, meagre individuals, with lack of muscular tone and relaxed abdominal walls, the problem to be settled is much easier, and we may indeed say, truthfully, "Liver is hard or liver is soft." Of course, we should never expect to find in hypertrophic cirrhosis the extreme *hardness* to tactile sensations which is characteristic of the atrophic form; nor is it rational to expect it when we consider the very great difference which exists in the variety of the fibrosis—fine in one case, coarse in the other. The same cause—alcohol—which produces fibrosis of the liver also effects interstitial changes in other abdominal organs, and particularly the kidneys, which are thus affected in a large proportion of cases.

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[1] Practice, fourth edition, p. 570.

[2] Ibid. cit., p. 209.

Despite the fact that it is affirmed that jaundice does not occur in atrophic cirrhosis, this statement cannot on inquiry and research be confirmed. On the contrary, it would seem as though we may and do have jaundice in all forms of cirrhosis. What is true is that jaundice occurs earlier in the hypertrophic form than it does in the atrophic, and hence, of course, when it does show itself in the latter form it is a sign of very bad augury. Many of the particular symptoms given by Hanot to designate specially his form of biliary cirrhosis are to be met with in the ordinary hypertrophic form, many of which are doubtless alcoholic; but when we come to consider the whole description of this writer we fail to find him in accord with other observers—mainly outside of France. Even the hyperplasia of bile ducts which Hanot considers so characteristic may be found in other forms. The jaundice does not always occur in the usual hypertrophic form, nor, again, is ascites always absent, and the spleen may not be visibly enlarged. Pain and pyrexia simply show advent of perihepatitis, and, although frequent in the hypertrophic form, it may also occur in other forms. Nervous phenomena and the typhoid state are more frequent in this form, but they are not absolutely distinctive. Chronicity is not constant, and, as we see, already, cases do not conform in all respects to a purely classical type. Where alcohol is a prominent factor in causation the symptoms approximate those of the atrophic form. No doubt in just such instances the hypertrophic liver is specially hard. The syphilitic liver, like the hypertrophic form, is often enlarged, extending in the right mammary line far beyond the right free margin of the ribs. If gummata are present they may be recognized, as already stated, by the distinct irregularities or bossing of the surface. If instead of gummata there is a cirrhotic process due to syphilis, the liver may also present a swollen appearance, and this is also true if amyloid degeneration has become manifest, which may be shown in advanced syphilitic conditions of the liver. In both syphilitic cirrhosis and amyloid liver the organ is hard and relatively smooth (Einhorn), and in the former case the fibrosis is said to be diffuse and monolobular. (Cheadle.) Not always is the syphilitic liver large; it may be small, and is thus often confounded with the alcoholic liver. This is especially to be regretted from a therapeutic standpoint, knowing as we do how invaluable antisyphilitic treatment is in these cases. Perihepatitis, while not pathognomonic of syphilitic liver, occurs very frequently in connection with it. Moreover, in this form a similar condition is more apt to extend to other organs, particularly to the peritoneum, where it causes that adhesive inflammation which of itself is quite characteristic. Where syphilitic cirrhosis exists it may be more or less advanced. Indeed, its presence may only be revealed by a slight scarring of the free surface and some thickening of the hepatic capsule. Such pathological conditions may serve to fix a diagnosis post-mortem where there has been no syphilitic history and where signs and symptoms during life left one in reasonable doubt as to the etiology of the case. Inasmuch as syphilis of the liver is a late form of the disease, I have known instances where the patient seemed almost oblivious of his ever having had it, and where he could only with some difficulty be made to understand any connection between his previous history and his actual symptoms. I am now having in mind educated and fairly intelligent men of the world. This is a practical observation of importance from the point of view of insistence upon antisyphilitic treatment at times, even though the patient is skeptical and disposed to resist or follow out his own impressions of his ailment. No organ at times becomes more important in the history of hepatic cirrhosis than the heart. As soon as it shows symptoms even of dynamic inefficiency immediately there is a tendency to venous stagnation, which must promote growth of fibrous tissue and development of ascites. Alcohol, as we know, affects the heart as it does other organs; but here it seems rather, as a rule, to produce fatty or other degeneration of the muscular fibres than deposits in interstitial tissues. No doubt these cardiac changes account for many sudden deaths in syncope, or sudden asthenia otherwise unexplained. Frequent stimulation may tide over some of these attacks, but to be effective it must be employed without delay.

I have already indicated the effect of alcohol upon many abdominal organs. Of course, we can understand if these organs are much implicated their influence upon the rapid march of hepatic cirrhosis is greatly felt. Hepatic cirrhosis is particularly liable to be complicated with acute miliary tuberculosis of some organs, and very many patients die from this rather than the hepatic disease itself. In this way it is shown how imperative it is for these patients to keep up good nutrition and live all the while nearly in the open air. The statements of many observers corroborate this. Thus Osler writes: "In seven of my series the patients died with either acute tuberculous peritonitis or acute tuberculous pleurisy."[1] It is curious, on the other hand, that distinguished and careful clinicians like Flint and Fagge apparently make no note of it.

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[1] Ibid. cit., p. 571.

The prognosis of hepatic cirrhosis has usually been most gloomy. Once ascites is revealed the patient's lease of life appears very limited. To-day we are more encouraged and regard certain cases very hopefully. Even in the advanced cases of atrophic cirrhosis we cannot always be sure to what extent some, not to say many, of the liver cells retain good functional power—at all events, power sufficient to help continue good nutrition if attending conditions be treated rationally.

It is recognized generally that we have no remedy which will make disappear the formation of fibrous tissue in the liver; but the ascites may be removed, even in these extreme cases, and allow the collateral circulation to act, with relief, to a certain degree, of the obstructed portal circulation. Then, every time effusion becomes considerable and organs are interfered with in their action through pressure, let paracentesis be repeated.

It has been shown by Fagge that cirrhosis often becomes quiescent before it reaches its final stage.[1] Flint seems to corroborate this view, because he says emphatically that life has lasted months and years after ascites had become developed and been recognized. Even in some atrophic cases of advanced type nutrition is still preserved, and this is always of good augury, because, despite the ascites, it shows some liver cells able to function normally. Of course, there is a possibility in these cases that we have not to do with a true cirrhosis of the liver itself, but with a perihepatitis or chronic peritonitis which was very probably the cause of the abdominal effusion. In this connection I would quote Fagge[2] again to corroborate my statement.

In hypertrophic and syphilitic cirrhosis the prognosis is much more hopeful. In the former case the development of fibrous tissue is of immature form, and occurs more rapidly with intercurrent conspicuous cell proliferation. This rapidly formed connective tissue is never so dense nor is it so destructive to the liver cells as that of the atrophic form of cirrhosis. In these cases there is often relative youth and good nutrition. Hence, from all these standpoints the prognosis is more favorable.

The prognosis of hepatic syphilis is even more hopeful. Of ten cases reported by Einhorn "only one patient died, and even this one had been considerably benefited for five or six months, whereas all the others were either perfectly cured or much improved."[3]

Of course, if cirrhosis could be recognized at an early stage, and before the advent of ascites, the outlook would not be immediately unfavorable. Sometimes we are able to make this diagnosis from certain prominent signs or symptoms: the indurated, large liver—an organ enlarged—and also irregular, vascular stigmata on the skin and face, and passing jaundice; an enlarged heart, constipation, dyspeptic symptoms, palpitations, and, it may be, some emaciation. We should act in accordance with the manifest indications, and among these is one primary and essential to correct absolutely—an alcoholic habit, if, as is usual, it has previously existed.

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[1] Paper read before British Medical Association, 1883, vol. ii. p. 566.

[2] Practice of Medicine, vol. ii. p. 815.

[3] Ibid. cit.

We often meet with combined cases of alcoholic and syphilitic cirrhosis. Some such cases have been known to recover during several years. This recovery was evident through subsidence of the ascites and recovery of the general health. (Cheadle.)

The first and most important indication of useful treatment arises from the point of view of arresting growth of fibrous tissue in the liver, and in this way the preservation of the remaining healthy liver cells. This is accomplished, if at all, by abandonment of the use of alcohol, which cannot be too urgently insisted upon in the great majority of instances. Of course, there may arise states of anæmia, prostration, or great weakness in which a moderate amount of good alcoholic stimulant may be imperatively required, at least for a time, and under such circumstances should surely not be withheld. This must be regarded merely as a temporary necessity, and we must have in view constantly the fact that it has been shown that even if fatty degeneration of liver cells be begun it will often be arrested if alcohol be wholly given up. A judiciously arranged dietary is only of secondary importance, and yet it is undoubtedly true that the digestive organs must be supplied with suitable food products, properly prepared, to sustain active nutrition and to ward off, as far as may be, certain functional or even organic disturbances in part dependent upon auto-intoxication of gastro-intestinal origin. Fats and sugars should be notably diminished in the dietary, for the reason that in the impaired liver functional power is decreased, and is shown especially in its inability for proper assimilation of foods containing many such elements. The simplest dietary of milk, eggs, stewed fruits, well-cooked fresh vegetables, and the lighter meats is of decided utility. Semmola's is the only rational treatment, viz., to reduce the quantity of food to a minimum and give it in a form which will tax the liver cells least. Milk is undeniably the best of all; but when this becomes intolerable, as it often does, eggs and other light food may be added. No doubt some instances of cirrhosis of the liver originate in auto-intoxication from the stomach and bowels. In all instances aggravation of the developed disease may result from it; hence over and beyond appropriate dietary the use of suitable antiseptic remedies like guaiaquin and benzosol (Gasper) may be formally indicated. Among drugs which have a decided action, and one most beneficial in almost all syphilitic cases, iodide of potassium easily ranks first. There can be no question, in view of very many recorded cases, that gummata in their early stages are reabsorbed through its action. It is highly probable, also, that perihepatitis and similar conditions of other abdominal organs (spleen, peritoneum) are very favorably affected. It is not a sufficient reason to proscribe the use of the iodide because the syphilitic nature of the case is not always perfectly clear. In many instances doubt may legitimately exist. Indeed, in many combined alcoholic and syphilitic cases a positive differential diagnosis as to how much is due to alcohol, how much caused by syphilis, is impossible. The only safe rule, then, is to give up alcohol and take the iodide. Again, in instances where there is no history of syphilis and no evidence discoverable of its presence, it is yet wise at times to administer the iodide, the reason being that purely alcoholic[1] cases and those due to syphilis of the nature of an interstitial hepatitis are often confounded one with the other.

In the event of evidences of heart weakness, and in view of the well-known post-mortem findings, digitalis and heart tonics should be employed judiciously. Salines and diuretics are of some use in the relief of ascites, but, as a rule, are of more value in warding off its return for a while after paracentesis than in preventing the necessity of this operation. The explanation of this is not perfectly clear, although a theory for it may be readily offered, viz., that the absorbents relieved from pressure are more efficient.[2] Very active purgation by the use of hydragogue cathartics is at no time indicated, as it is useless and may become directly injurious by depleting the patient's strength very much or by bringing on diarrhœa often difficult to arrest. On the other hand, the moderate use of mineral waters to keep the bowels regular may be very necessary to the patient's welfare.

In the treatment of hypertrophic cirrhosis Nothnagel has wisely insisted upon small, repeated doses of calomel. Vaughan believes that later this form of disease will be treated mainly by surgery, as it is the only way by which the gall-bladder and biliary vessels can be disinfected.

Billings quotes Kussmaul as using chloride of ammonium to prevent proliferation of the connective tissue cells, and he (B.) claims in this way he has seen induration of the liver disappear as well as some local symptoms connected with the disease.

Whatever may have been the objections in former years to paracentesis, legitimate or unsupported, to-day it seems very clear that in early and repeated paracentesis the best hope of the patient lies. Even in advanced instances of atrophic cirrhosis life is prolonged and suffering diminished; but in instances where we have an enlarged liver with ascites, which in such cases often occurs when the disease is not far advanced, we get our best results from paracentesis. The fluid may recur in smaller quantity after each paracentesis, and in some undoubted examples, after several operations, no fluid has recurred, and so far as symptoms are concerned, we may properly say a recovery has taken place. This may last for many years. I have had at least one such instance in my practice. The patient recovered from the ascites, and subsequently died of another disease than hepatic cirrhosis, as was evident at the autopsy. The danger to-day of peritonitis arising from paracentesis is very slight, indeed, if ordinary aseptic precautions be observed. I do believe it is wiser to employ the ordinary aspirator or Flint's modification of the Davidson syringe, than the trocar and canula, and mainly because the flow of fluid is easily regulated, and thus there is less danger of syncope or heart failure, which might be directly caused by too rapid evacuation of fluid where cardiac degeneration is present. This accident may also be obviated, as we know, by the prompt use of a binder as the fluid flows from the abdomen. The binder also is useful in delaying return of ascites. There is less risk of serious injury to the intestines with the small needle attached to the aspirator than with a moderate-sized trocar.

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[1] Of the frequency of this form, one may judge from Flexner's report of autopsies done at the Philadelphia Hospital. Among eighty-eight cases of syphilis of the liver, forty had interstitial hepatitis.—Medical Record, October 19, 1901.

[2] Thromboses of the portal vein may cause rapid reaccumulation of ascites.

Unfortunately, paracentesis is not always thoroughly satisfactory. The canula may become blocked in different ways and for different reasons, not always easy to obviate. In our very desire to benefit our patient and withdraw most of the fluid from the cavity we may run the risk of wounding the intestine. Sometimes the cause of the obstruction of the canula is a piece of false membrane; again it seems to be the intestinal wall, and we must be careful not to injure it. In some instances where we expect to obtain a large quantity of fluid we really get very little, which is discouraging both to patient and physician. Occasionally it seems as though the posture of the patient was responsible for this, but I have found the difficulty to arise when the patient was sitting up and also when the patient was lying down.

Some authors have objected to paracentesis because, they say, after it the ascites recurs more rapidly, and that, besides the risks alluded to, it is very depressing through loss of albumin from the blood on every occasion it is performed. This objection has little or no value. as a rule, especially if the patient be in fairly good general condition, Of course, if the patient is very old and feeble, or the disease very advanced, paracentesis is likely to hasten the fatal termination. On the other hand, where the abdominal and thoracic organs have been

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[1] British Medical Journal, 1883.

compressed or pushed away from their normal position by excess of abdominal fluid there is no question that the fluid should be speedily removed. Thus great distress is often relieved, the kidneys begin to function with renewed activity, and the general condition of the patient is notably improved. The lungs are enabled to breathe freely again, and the heart becomes regular and fails to intermit. While getting additional power very rapidly, cough, expectoration, hypostasis, and even pleuritic effusion due to upward pressure soon disappear. Sometimes after paracentesis there is more or less leakage from the abdomen through the hole made with the needle or trocar. This is often very annoying to the patient, as his clothes and bedding become thoroughly wet; and yet it may be useful in a few instances from the fact that the drainage continues for several hours after the operation is concluded, and from regions the needle could not reach. In this connection it is proper to refer to continuous abdominal drainage by means of a permanent canula left in the abdomen after the trocar is withdrawn. I performed this operation on one occasion, with apparently some good result; but the dangers from aspiration of air into the cavity, peritonitis, and suppuration are such that I felt later that better ultimate results could be expected from repeated puncture and before the fluid was permitted to reaccumulate in too large quantity, or caused any notable distress, or interfered much with the healthy function of any important organ.

"The removal of fluid by continuous drainage has been practised with some success by Dr. Caille and Dr. Elliot in America, and by Dr. Urso in Italy."[1] And certainly in some cases it would appear to be safer and quite as effective as the radical operation to produce new anastomoses.

On the other hand, Weir,[2] who used permanent drainage in connection with or rather subsequent to the radical operation, "would hereafter prefer to resort to paracentesis if it became necessary, as the risk seems less."

It is surprising at times to remark the powerful diuretic effect of paracentesis. Kidneys that previous to it were inactive become active, urine that was albuminous, of high specific gravity, and contained casts accompanied by uræmic symptoms, has changed remarkably. The flow of urine has wonderfully increased in quantity, and its characters have become almost normal, while all uræmic evidences have soon disappeared. Without the paracentesis both purgatives and diuretics seemed wholly worthless to produce these much-desired effects.

If we wish to get the best effects from repeated tapping we must have patients under our immediate care, so that they may be tapped again when the proper moment arrives. Tapping should be repeated in proper cases until fluid ceases to reaccumulate. During this period it should be our effort to maintain the patient's nutrition to the highest degree possible. Through constant care and attention we obtain the best results; through carelessness or permitting the patient to absent himself too long from observation we have often cause for regret, and the patient relapses into an impoverished and threatening state.

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[1] Cheadle, p. 88.

[2] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1899, p. 723.

On the other hand, Hale White's observations would go to show "that in the cases of cirrhosis of the liver with chronic peritonitis the survival for a considerable length of time was due to the fact that the latter lesion assists in some way in the formation of a collateral circulation."[1]

Now, as Osler writes: "We know that extreme grades of contraction of the liver may persist for years without symptoms when the compensatory circulation exists. The so-called cure of cirrhosis means the re-establishment of this compensation."[2]

Upon this idea of re-establishment of this compensation rests the modern radical surgical treatment of cirrhosis of the liver. The method is to promote adhesions between the liver, spleen, and abdominal walls and diaphragm, thus helping collateral circulation by new vascular channels. This is attempted, first, by completely evacuating the abdominal contents, and then, by thorough scrubbing of the organs referred to, set up a certain amount of irritation which shall tend to make the formation of new vessels more probable. In this connection Cheadle is of the opinion that if we consider the conditions where the new vessels are found naturally they do not seem favorable, being associated with the worst cases, and do not lead one to try to produce them artificially. Opposed to this view we would cite three cases cured: one of Osler's,[3] operated on by Dr. Bloodgood at Johns Hopkins Hospital; another of Brown's,[4] another of Frazier's.[5] In one instance under my care, operated on by Dr. F. H. Markoe, nearly two years ago, the patient has had recurrent ascites, necessitating repeated tappings since the radical operation. Two of Osler's cases were unsuccessful. As regards one or two tappings after the operation, this we should expect where a drainage-tube has not been introduced, the reason being that time is required for the formation of adhesions and new veins; and the introduction of the drainage-tube is now shown to be bad from a surgical standpoint, as it opens a channel of infection. (Frazier.)

In Dr. Brown's successful case, at first referred to in the *Presbyterian*

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[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1901, p. 259.

[2] Ibid. cit., p. 566.

[3] Ibid. cit., p. 577.

[4] New York Medical Record, October 19, 1901, p. 637.

[5] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, December, 1900, p. 661.

Hospital Reports for 1900, and the patient subsequently presented at a meeting of the New York State Medical Society, held October 16, 1901, at the New York Academy of Medicine, the condition was one of atrophic cirrhosis, due to alcohol. There was no syphilis and no malaria, although the spleen was enlarged. There was no pronounced venous engorgement on the abdominal walls. The patient was operated upon over two years ago. There has been no recurrence of ascites, and the nutrition remains good. The patient is doing his usual work—that of a day laborer.

Frazier[1] in the remarks he makes, prompted no doubt by a successful case, wisely insists, as a *formal* contraindication to the operation, upon the absolute lack of functional power in the liver cells—*i. e.*, shown usually by extreme atrophy. (Packard.) In his judgment, the presence of cardiac and renal degeneration is only a relative contraindication. He also states that it is essential, in order to avoid fatal toxæmia, that the collateral circulation should be formed gradually. Where the cases have been carefully selected he " believes the operation has a future."

Fourteen cases are thus far reported, seven of which appear to have been materially benefited or cured by the operation (Brown) and three died from the operation.[2] " The many instances of practical cure following tapping, and the uncertainty of the exact pathological state of the liver, together with the somewhat formidable character of the operation and liability of infection, invest the operation at present with a large element of doubtful expediency."[3] These remarks, cited textually from Dr. J. D. Bryant's work on *Operative Surgery*, appear to me eminently wise.

In all these cases, moreover, it is judicious to properly estimate the amount of functional disturbance, if possible, of the liver cells (icterus, acholic stools, urobilinuria, etc.) as well as the physical changes of the liver itself. In it may be found, as Brown says, a formal contraindication to the wisdom of the operation. Neumann[4] also wisely insists " that the liver cells be not too greatly impaired in their functional capacities, and that every precaution should be taken in selecting proper cases, in order not to permit discredit to fall upon so valuable an operation."

Brown " feels assured that the great risks attending operations on advanced and failing cases will be notably wanting in similar procedures applied in earlier stages of the disease."[5] No doubt this statement is correct, but in my judgment it is correct only so far as it applies to all important or dangerous surgical procedures. The great difficulty is to convince the pure physician with the facts as reported up to date, and allowing for errors of diagnosis with respect of the precise nature of the liver condition and for the concomitant conditions almost always present (heart, kidneys, spleen, pancreas, etc.) that such a stand is justified. I am scarcely of that opinion until it be shown that the radical operation in similar instances gives better results than the far less serious procedure of simple and repeated paracentesis.

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[1] Loc. cit.

[2] Later Packard reports twenty-two cases, with nine recoveries and eight deaths (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1901, p. 265), and in a " note " one death—one doubtful.

[3] Bryant, Operative Surgery, 1901, vol. ii. pp. 802, 803.

[4] Quoted by Brown (p. 11).

[5] Page 16.

Dr. Weir's standpoint appears to be a more rational one, viz., "The operation was worthy of trial in apparently hopeless cases of liver cirrhosis in which the abdomen rapidly refilled after repeated tapping, and in which the large quantity of the fluid was producing fatal exhaustion."[1]

Dr. McBurney in discussing Dr. Weir's paper thought that analogy with what occurred after simple incision in cases of tuberculous peritonitis allowed one to question whether the relief of ascites in cirrhosis of the liver which followed the radical operation was brought about by the development and growth of vascular anastomoses. This view of McBurney's seems to approximate that of Dr. Osborne, of New Haven, and of Dr. Cheadle, already cited.

On the other hand, Packard and Le Conte[2] claim that "one of the ways by which repeated tappings may aid in the recovery of cases of cirrhosis of the liver is through the formation of adhesions similar to those aimed at in the operative procedure, but to a less degree." Be these views as they may, I cordially assent to their previous statement,[3] that "these cases of advanced cirrhosis of the liver, operated on or not, die with evidence of progressive toxæmia and gradual failure of all the organs to do their proper work."

Despite these views, they state in their conclusions, as their opinion, "that where the diagnosis of pure portal cirrhosis of the liver can be made, and where persistent and well-directed medical treatment is productive of insignificant results, the operation should be strongly recommended."

Here, it seems to me, the great difficulty lies in this precise diagnosis at the present time; for do we not know that constantly our autopsies show that concomitant lesions exist which render nugatory all radical operations—*i. e.*, other kinds of cirrhosis, chronic peritonitis, advanced degeneration of other organs, like the heart and kidneys, etc.?

There can be little doubt that in very many cases of hepatic cirrhosis it is important to do what we can medicinally and otherwise to keep the heart in a vigorous state. Once it becomes inefficient functionally, either because of simple dynamic weakness or owing to structural changes, the development of the ascites is certainly more rapid and more apt to recur. We recognize in this way that the obstructed portal circulation due to fibrosis is also heightened notably by venous stagnation caused directly by impaired heart power. No doubt the explanation of instances of early effusion in hypertrophic cirrhosis is thus often satisfactorily given. If such a view be admitted it can be readily understood why we can obtain good effects from suitable treatment in just such cases. An important part of this treatment must be the maintenance of sufficient cardiac power if it be possible to prevent venous stagnation in the liver.

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[1] New York Medical Record, January 28, 1899, p. 143.

[2] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1901.

[3] Ibid., p. 257.

In instances where venous stagnation occurs of cardiac origin the hepatic veins at the centre of the lobules are especially affected, and it is also true in these instances that here is likely to be formed the fine fibrous deposit which, as we have seen, characterizes hypertrophic cirrhosis. Not always is the cardiac inefficiency shown by evident physical signs, nor, indeed, by the presence of ascites; still, in hypertrophic cirrhosis or simple engorged liver it is a safe plan to give, for a while at least, small, repeated doses of digitalis, and note its obvious effects either on the size of the liver directly or some of the suggestive symptoms connected therewith.

Appended are histories of cases of "omental anastomosis."

CASE I.—Case of cirrhosis of the liver in which radical operation was performed by Dr. F. H. Markoe. J. S., aged fifty years, married; hotel steward; born in the United States; admitted to St. Luke's Hospital June 30, 1898. Alcoholic history; suffering from ascites. Liver probably enlarged; rough and nodular. Abdomen much distended; evidences of fluid; superficial veins enlarged.

First Aspiration. Three days after admission, 125 ounces obtained; second: eleven days later 128 ounces obtained.

Treatment nil. Left hospital July 22, 1898; re-entered, December 6, 1898.

Abdominal fluid increased in quantity as compared with quantity before the last tapping, July, 1898.

Third Aspiration. Two days after admission, 370 ounces obtained; fourth: five weeks after admission, 400 ounces obtained; fifth: seven and a half weeks after admission 440 ounces obtained; sixth: ten and a half weeks after admission, 475 ounces obtained.

Treatment. Digitalis, iodide of potash, theobromine, copaiba, etc. Left hospital March 1, 1899, and readmitted two weeks later; abdomen greatly distended.

Seventh Aspiration. Day after admission; 390 ounces obtained. Left hospital, and readmitted April 17, 1899.

Eighth Aspiration. Day admitted; 426 ounces obtained.

May 13th. Ninth: 390 ounces obtained.

June 14th. Tenth: 450 ounces obtained.

July 10th. Eleventh: 330 ounces obtained.

August 12th. Twelfth: 452 ounces obtained.

October 7th. Thirteenth: 520 ounces obtained.

November 14th. Fourteenth: 510 ounces obtained.

Treatment. Codeine for cough.

January 22, 1900, Dr. F. H. Markoe performed upon the patient the operation of "omental anastomosis," and 500 ounces of light yellowish fluid slowly evacuated from the abdomen. On palpation, the liver was found large; also some abdominal adhesions. Vomiting of green fluid off and on for six days following operation.

Fourteenth Aspiration. Six weeks after operation; 138 ounces obtained.

Subsequently, up to November, 1901 (twenty-two months later), patient has been aspirated about fifteen to seventeen times, which, with those performed before operation, make about thirty in all.

November, 1901. He is now confined to bed, and is unable to leave it by reason of asthenia.[1]

Case II.—A second case of "omental anastomosis" for cirrhosis of the liver was performed by Dr. F. H. Markoe at St. Luke's Hospital, April 27, 1901. The patient had marked ascites; was a boy, aged thirteen years, and had been quite a drinker. Subsequent to the operation the patient was tapped once before he left the hospital, on May 21, 1901. Since that date he has returned about every six weeks to have the fluid removed from the abdominal cavity. The patient's general condition is about the same as before the operation. The ascites is probably somewhat less.

Case III.—The following case of "omental anastomosis" is one in which Dr. F. W. Murray operated.

History. P. A. B., married, aged forty-eight years; no specific disease; excessive alcoholic habits.

Four months before admission to St. Luke's Hospital patient noticed yellow conjunctivæ and loss of flesh and strength. His abdomen increased by eight inches in size. This increase subsided for a time under calomel and salines, but soon returned. April 2, 1900, entered hospital; 328 ounces of fluid removed by tapping; largely reaccumulated in two or three days. Transferred to surgical service April 18th.

Abdomen measured 44½ inches at a point three and a half inches above the umbilicus; percussion flat over the whole abdomen. Operation April 19th, by Dr. Murray. Incision four inches long at the left of the umbilicus; 3¼ gallons of fluid removed; omentum thickened and vessels injected; sutured to parietal peritoneum by five interrupted sutures of No. 1 catgut; stood operation well. Urine acid; specific gravity, 1020; slight trace of albumin; no sugar; few hyaline casts.

Aspirated April 23, and 64 ounces of fluid obtained. April 30th, circumference 39 inches. May 2d, primary union of wounds. May 12th, œdema of feet; stuporous; pulse rapid; coryza marked; erythema over nose, diagnosed as erysipelas. May 13th, temperature between 98.3° and 99° F. Pulse varies from 90 to 100. Respiration, 24. Died. (Above history furnished by Dr. Martin.)

Note.—Since writing the foregoing paper a valuable contribution from the pen of Dr. George E. Brewer, on "The Surgical Treatment of Ascites Due to Cirrhosis of the Liver," has been published. From it we take the report of "5 personal cases and analyses in tabular form from about 50 more from the literature." From reviewing these statistics Brewer finds at least 6 patients who have been cured of ascites by this procedure—*i. e.*, Talma-Morrison operation—and who have remained well for two years or more; 6 others have died, with relief of this symptom from two to six months before death, or who had not been under observation long enough to demonstrate a permanent cure. Another patient suffering from hemorrhage of the alimentary canal was promptly cured by this operation. Many others have been materially benefited; 38 have recovered from the operation; and, considering that the great majority of these were within a few weeks of inevitable death, he thinks that it should encourage us to suggest operation at an earlier and more favorable stage of the disease. If this suggestion is followed he believes that statistics will show a great improvement over those he is able at this time to present."[1]

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[1] Patient died December 15, 1901, from heart failure.

In a later contribution on this subject Dr. W. Murrell, of Westminster, writes[2] that "much depends on careful selection, and the best results would probably be obtained in the pre-ascitic stage when the diagnosis rests on the alcoholic history, with hæmatemesis and enlargement of the liver and spleen."

A CASE OF MULTIPLE MELANOTIC SARCOMA UNSUCCESSFULLY TREATED BY X-RAYS.

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The X-ray treatment of various malignant diseases has been used long enough to thoroughly establish the great utility of the method in certain cases and locations. For the ordinary slow-growing epitheliomata of the skin the X-rays are practically specific if the treatment is instituted before glandular involvement has occurred. In my own cases I have not failed to apparently entirely eliminate this disease in from eight to twelve sittings of ten minutes each—I say apparently, because the time elapsed has not been sufficiently long to call the condition a cure; and the X-ray therapeutist should be willing to apply the same three-year rule to his work as does the general surgeon.

But however firm, therefore, our conviction that the X-ray is of inestimable value in the above-mentioned conditions, there is an equally strong feeling that certain malignant tumors have been made

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[1] Journal of the American Medical Association, February 22, 1902, p. 135.

[2] Lancet, June 7, 1902, p. 1604.